

# Individualized Exercise Interventions for Spinal Pain

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### **Individualized Exercise Interventions for Spinal Pain**

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## Individualized Exercise Interventions for Spinal Pain

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## **ABSTRACT**

Exercise is the most effective treatment for the management and prevention of spinal pain, yet on average it delivers small to moderate treatment effects, which are rarely long-lasting. This review examines the hypothesis that outcome of exercise interventions can be optimized when targeted towards the “right” patients and when tailored to address the neuromuscular impairments of each individual.

## **SUMMARY for TOC**

Individuals adapt differently to spinal pain and exercise outcome can be optimised when tailored to each individual.

## **KEY POINTS**

- Spinal pain is a leading cause of years lived with disability, with massive associated socioeconomic costs. More than half of those affected by an acute episode of pain still report pain and disability one year later.
- Exercise is a common and effective treatment for spinal pain. Yet current exercise programs rely on a “one size fits all” approach and usually fall short of success.
- Studies have revealed the complexity and individual variability of the neuromuscular adaptations accompanying pain, and the heterogeneity between patients with respect to the contribution of physical features to their chronic pain disorder.
- We provide evidence supporting the hypothesis that outcome of exercise interventions can be optimized when targeted to the “right” people and towards the neuromuscular impairments that present for the individual.
- An exercise approach, based on identification of patient-specific tailored interventions, has the potential to dramatically improve outcomes.

## **KEY WORDS**

Exercise; spinal pain; rehabilitation; motor control; personalized rehabilitation

## INTRODUCTION

Low back pain is the leading cause of ‘years lived with disability’ (3). The incidence of low back pain has reached epidemic proportions, affecting up to 84% of adults at least once in their lives (4). Neck pain ranks 4<sup>th</sup> as a cause of ‘years lived with disability’ (3) and has a 12-month prevalence of 30% - 50% (24). Both conditions tend to be persistent or become recurrent; up to 85% of people can expect some degree of ongoing pain for many years after their first episode (1, 26). Collectively, low back and neck pain are associated with massive socioeconomic costs, including the cost of medical treatment, work absence, and long-term consequences including decreased ability to perform activities of daily living. The enormous indirect socioeconomic costs due to chronic pain exceed those estimated for heart disease, cancer or diabetes (16).

Spinal pain often originates from sources that are not readily identifiable and frequently there is a mismatch between objective findings of structural abnormalities and symptoms. Features of spinal degeneration such as facet joint osteoarthritis, intervertebral disc space narrowing, spondylolysis, and spondylolisthesis are equally common in those with and without spinal pain (29). As treatment cannot typically be directed towards pathological abnormalities, the alternative has commonly been directed towards generic approaches for symptom relief, functional improvement, return to work, and the development of coping strategies.

Management of spinal pain is difficult and many established interventions have limited efficacy. Conservative intervention, particularly exercise, is the cornerstone of management of spinal pain. Effective and early management of pain and neuromuscular function via exercise is promoted as a critical element of management for spinal pain (19), recommended by clinical practice guidelines internationally. Yet, most conservative treatments for non-specific pain show small to moderate effects on average with little evidence of superiority of one treatment over

another (19, 30, 35). Importantly, these minimal gains are rarely maintained in the long term (42). There are several factors, including lack of adherence to therapeutic recommendations during and subsequent to treatment, that mitigate against pain relief. This poor outlook is not unique to conservative treatments; it is the case for all current treatments including surgery and pharmaceutical interventions. New approaches are needed.

An evolving hypothesis, that is biologically plausible and founded on recognition of the obvious heterogeneity within the population with spinal pain, is that exercise is likely to be most effective if tailored for the individual. The alternative “one size fits all” approach to spinal pain usually falls short of success (19, 30, 35). There is growing evidence of individual variation between patients that is likely to be meaningful for exercise prescription, and emerging evidence of improved outcomes from tailoring interventions. There are likely to be two layers required for this approach. First, exercise can impact spinal pain in multiple ways; from modification of tissue loading to reduce nociceptive input, to augmentation of physical fitness, and exposure to movement to reduce threat. These approaches will have different effects for different patients. Second, if tissue loading from suboptimal movement is considered important, it would be expected that a tailored approach would be necessary to address the specific features of loading (e.g. muscle activation, posture/alignment, and movement strategy) of relevance for the individual.

In this review, we focus on the role of physical factors, in particular deficits in neuromuscular function, in development and transition to persistence of pain. We provide evidence of variability in motor and sensorimotor function in the presence of clinical pain, and variation in the response to acute nociceptor input. We also present evidence that illustrates variability in response to standardized exercise programs in people with spinal pain, with some

people gaining complete relief of pain and others having no or only marginal benefit. Together these data offer support for the hypothesis that outcome of exercise interventions can be optimized when targeted to the “right” people and then towards the neuromuscular impairments that present for the individual (Figure 1).

## **SPINAL PAIN: A HETEROGENOUS CONDITION**

Spinal pain forms a heterogeneous group with variable and often complex patterns of co-existing biological and psychological features. Biological and psychological features interact to drive central sensitization processes that amplify input and processing of nociceptive input and pain, or maintain pain in the absence of on-going input from the tissues. Social factors may also amplify the pain experience. Thus, assessment and management of people with spinal pain disorders must always be considered within a biopsychosocial framework (Figure 2A). Weighting of biological, psychological and social factors will vary across individuals (Figure 2B). Some patients present with largely biological factors including physical features that load their tissues suboptimally leading to on-going nociceptive input that continues to drive and maintain their chronic pain disorder. Such physical features include suboptimal posture/alignment, movement and altered patterns of muscle activation (Figure 2A1). In this case, physical interventions including exercise which targets the provocative motor behaviours will have greater potential for relevance within the rehabilitation program than it would for a patient whose pain is maintained purely by central processes. In contrast, other patients present with dominant psychological features such as pain catastrophizing, fear avoidance, anxiety, depression or stress which outweigh the physical factors (Figure 2A2) and in these cases the relevance and benefit from specific attention to changing the way that the patient moves will likely be less, but they may benefit from exercise that provides experience with healthy

movements to ameliorate fear and address deconditioning. If social factors such as poor support or high job demands with low reward are amplifying the patient's perceived pain (Figure 2A3), again physical interventions will take less priority. Considering the complexity and heterogeneity of spinal pain presentations, usually multidimensional and multidisciplinary approaches are warranted. Here we focus on neuromuscular changes, especially in relation to individual variability, and the contribution of exercise to an overall management programme for spinal pain when tissue loading remains a relevant biological element of the patient's presentation.

## **MOTOR AND SENSORIMOTOR ADAPTATIONS IN CLINICAL SPINAL PAIN**

Differences in motor and sensory systems between patients with low back or neck pain, and pain-free controls have been highlighted in extensive literature, and could both precede and follow the onset of injury/pain (21). The literature is characterised by enormous variation, with few studies highlighting similar features, partly explained by the huge variation in experimental methods and populations tested, and partly explained by substantial redundancy in motor control of the spine, with multiple muscles available to perform a specific action. Many studies adopt a simple approach that aims to identify features that differ between individuals with and without spinal pain. This approach has revealed some observations that are relatively consistent across individuals. Yet many differences in neuromuscular control are not consistent and have been revealed primarily by comparison of *a priori* defined subgroups.

Adaptations that appear relatively consistently in many individuals with spinal pain include delayed/reduced activation of the deeper back muscles (transversus abdominis e.g. (23), multifidus e.g. (32) in low back pain). Conversely, activation of the oblique abdominal muscles, and some components of the erector spine muscle group, is often augmented e.g. (39). These relatively consistent changes are accompanied by changes in brain organisation characterised by



differing size and location of cortical representations and convergence of brain representations for multiple muscles (43). Taken together these observations imply a change in strategy, potentially representing a shift in motor strategy from one that involves discrete activation of muscles to fine-tune intersegmental motion (a function to which the deeper trunk muscles can contribute via their direct segmental attachments akin to a muscular sleeve around the spine); towards one of stiffening that would be achieved by activation of the more superficial muscles. This shift would likely have an impact on the quality of tissue loading, and lead to potential for both poorly controlled intervertebral motion (from poor activation of the deeper muscles) and excessive compression (from augmented co-contraction of larger muscles), depending on the net consequence of the adaptation.

In a similar manner to the lumbar spine, there is evidence for individuals with neck pain that activation of the deep cervical flexor muscles is less than for pain-free individuals e.g. (12). Greater activity of the sternocleidomastoid and anterior scalene muscles in patients with neck pain than pain-free individuals has also been reported in multiple studies, during multiple tasks and across several cervical spine disorders including cervicogenic headache, idiopathic neck pain, whiplash associated disorders and work-related neck pain suggesting that it is a common feature in patients with neck pain disorders (28). Collectively this indicates a reorganization of the motor strategy to perform cranio-cervical flexion.

Although the preceding examples highlight some changes in motor function that are common amongst people with pain, and this could be used to defend the appropriateness of a more systematic application of exercise as treatment, these changes are not present in all individuals and are generally combined with other features that are more variable. In most studies variation has been revealed simply as greater standard deviation of motor and sensory measures when patients with non-specific spinal pain are compared with pain-free controls (e.g.

(10)). Other studies have purposefully selected specific subgroups of patients and identified key differences that can be directly interpreted with consequences for tissue loading. When again considering the example of reduced activation of the deep cervical flexor muscles in people with neck pain, an evaluation of individual data revealed that some patients with neck pain demonstrated activation levels which were consistent with that of asymptomatic people, despite that, on average, the patient group showed lower values of muscle activation (12). It is understandable that patients with no deficit in deep neck muscle activation would be unlikely to benefit from an exercise intervention such as cranio-cervical flexion training which aims to facilitate the activation of the deep cervical flexor muscles.

There are numerous examples of individual variation in profiles of tissue loading between spinal pain patients with respect to posture/alignment and movement. An example related to posture/alignment comes from subgroups of low back pain patients who sit with the spine more flexed or more extended (5), with associated differences in muscle activation. These opposite presentations imply opposite targets for treatment to optimise loading. From the perspective of movement, numerous features have been identified. For example, in a specific subgroup of patients with back pain, motion of the hip is accompanied by earlier and greater motion of the lumbar spine and pelvis, with implications for bias of tissue loading to the spine rather than sharing between the hip and spine (46). There are many other examples. These observations provide clear direction for tailoring of exercise to the individual; to target the specific feature of neuromuscular control related to suboptimal tissue loading. Such obvious targets for training are not present for all patients, and a greater understanding of the link between nociceptive input and changes in sensorimotor features has required detailed examination with experimental methods, including experimental pain.

## **INDIVIDUAL VARIATION OF MOTOR ADAPTATIONS TO DISCRETE NOCICEPTIVE INPUT**

A common approach to study the effect of nociceptive input on neuromuscular control has been to evaluate the sensorimotor response to a discrete experimental noxious input. Although not replicating all aspects of clinical pain, this approach enables: identification of the response to nociceptive afferent discharge in the absence of tissue injury; clarity regarding changes that follow rather than precede pain; response to a stimulus that involves a similar mechanism and location for all participants; and a sensitive approach to study subtle changes in muscle activation between painful/non painful conditions. The characteristics of pain share some features with clinical pain and several studies show similarities in the way motor control is changed e.g. (9).

As reviewed above, clinical pain may alter the task-related modulation of muscle activity so that neuromuscular control of the spine to achieve a specific task objective is solved by alternative combinations of synergistic muscle activities. The notion that nociception may induce a stereotypical motor response has been supported by some experimental data. For example, previous studies that used multi-channel (high-density) surface electromyography (EMG) to record the distribution of upper trapezius muscle activity before and during experimentally induced muscle pain, while maintaining a steady 90 degree shoulder abduction position, provided evidence of a relatively greater reduction in muscle activity in the cranial than caudal region of the muscle (7). Interestingly, the adjustments to noxious stimulation of the upper trapezius were consistent and were confirmed to be independent of the location or intensity of the painful stimulus i.e. the greatest reduction of EMG amplitude occurred in the cranial region of the upper trapezius muscle, even when nociceptive afferents in the caudal region were stimulated (8)(Figure 3). This finding implied that nociception induced a stereotypical motor response regardless of

pain location. Similar observations have been made from comparison of the effect of noxious input to the medial and lateral calf muscles (25). By more specifically evaluating adjustments in the behaviour of motor units located in different regions of the upper trapezius to experimentally induced pain, we recently confirmed differential changes depending on the region of the muscle which were not dependent on pain location (6). These findings indicate that nociceptive synaptic input is distributed in a non-uniform way across regions of the muscle and that the adjustments to pain were similar irrespective of the location of pain, suggesting a fixed motor response to pain anywhere in the upper trapezius. One interpretation of the adaptation is that it may aim to protect the cranial region from overuse since this region has higher pain sensitivity (6). This observation supports the notion that some aspects of neuromuscular adaptation to pain may be consistent between individuals.

Other recent data from studies of gait provide similar conclusions (45). When a noxious stimulus was provided to either the back (paraspinal muscle) or the leg (calf muscle), there were minimal changes in the muscle synergies for weight acceptance and push off, despite involvement of the painful calf muscles in those synergies (Figure 4). In contrast, the synergies involved in trunk movement and control were modified, again, regardless of the location of pain. The specific muscles and the degree of modification varied between individuals. These data support the notion that adaptation to pain appears to affect some aspects of motor function more consistently; noxious input at different sites led to preferential adaptation of the synergies that control functions secondary to locomotion with limited impact on synergies critical for task performance (weight acceptance and push off). However, there was some variation between individuals in how the adaptable synergies changed. (i.e. individual-specific flexibility). Thus, adaptation to pain is characterised by both invariance and variance, depending on the motor feature and the task assessed.

Drawing from the concept of variation in adaptation between individuals we have combined modelling and empirical approaches to study adaptation of the redundant trunk muscle system (22). In response to hypertonic saline injection into a back muscle during a slow trunk movement in healthy individuals we showed substantial variation between individual patterns of responses in recordings from 12 muscles. The pattern of increased, decreased and unchanged muscle activity differed for all participants. However, when data were included in an EMG driven model, the net outcome was enhanced stability (Figure 5). This observation highlights that uniform application of exercise would be inappropriate to modify loading strategy and an individualised approach would be necessary.

Again turning to the neck, when evaluating changes across synergistic muscles, evidence indicates that people show that some features of motor adaptation to noxious stimuli are highly variable. For instance, noxious stimulation of the splenius capitis muscle in pain-free volunteers triggered a subject-specific redistribution of muscular activation; in some participants the activity of a given muscle increased, whereas in others it decreased during pain (17) (Figure 6).

Variability in the response to nociceptive input likely relates to a number of factors including an individual's anatomy/biomechanics/anthropometry, the individual's habitual movements and postures, prior experience with pain, interactions between the nature and extent of the injury, the magnitude of pain and disability, and the presence and magnitude of attendant psychosocial/cognitive factors. Regardless of the cause, such variability likely contributes to the inconsistent symptomatic benefit experienced by patients following standardized exercise programs with responses ranging from an excellent outcome to minimal or no benefit when we consider that the exercises may be targeting features of neuromuscular function which are not affected in every patient or are not the main features driving the patient's pain experience.

## **EXERCISE FOR THE MANAGEMENT OF SPINAL PAIN**

Exercise is one of the most frequently recommended treatments for patients with spinal pain. Evidence from randomized controlled trials and systematic reviews demonstrates that exercise is effective at reducing pain and improving function in the treatment of chronic low back (41) and neck pain (18). This evidence is reflected in National and International guidelines for chronic spinal pain which consistently recommend exercise therapy. Exercise has been proposed to improve motor control, strength, endurance, flexibility, range of motion, general fitness as well as to improve mood and alleviate depression (41). Currently, there is no clear evidence that one particular type of exercise is more effective than another to manage patients with spinal pain (40). For instance, significant reductions in pain and disability have been observed for various types of training programmes in patients with neck or low back pain including motor control training and resistance training. Moreover, a recent systematic review revealed that no statistically significant differences were found for pain and disability between physical and behavioral/psychologically intervention groups in the medium- and long-term (38). Although, how and when certain types of exercise are most valuable likely depends upon when they are delivered. Exercise is also known to be beneficial for the prevention of spinal pain. A recent systematic review found evidence for both exercise alone (35% risk reduction for a low back pain episode and 78% risk reduction for sick leave) and for exercise and education (45% risk reduction for a pain episode) for the prevention of low back pain up to one year (42). Despite the statistically significant effects of exercise, systematic reviews of exercise for the management or prevention of spinal pain show small to moderate effects on pain and function, and the effects are rarely long-lasting (30, 36, 42). Moreover, positive effects of exercise have been shown to be most evident when compared with minimal interventions, placebo, or waiting list control groups (38).

One likely explanation for limited effect-size from trials of treatment and prevention of pain is that a “one size fits all” approach to exercise is inadequate and that assessment-driven targeted interventions are required to achieve meaningful and long-lasting change. Most randomized controlled trials have not adequately dealt with the multidimensional nature of chronic spinal pain (38). The expected benefit of exercise should consider other features of the patient’s presentation, which may impede positive exercise outcomes. As an example, consider Figure 7. This figure presents the percent reduction in neck pain intensity reported following a rehabilitation programme, including neck exercise, for individuals with chronic whiplash associated disorders (WAD) and idiopathic neck pain. Note a 47% reduction in neck pain after six weeks of exercise in the group of patients with mild/moderate idiopathic neck pain (data from (11)). An eight week rehabilitation programme, including the same exercises, resulted in a 37% reduction in neck pain intensity in people with WAD with signs of mechanical hyperalgesia (data from (27)). The response to the same intervention was only a 16% reduction in people with WAD with signs of widespread mechanical and cold hyperalgesia (suggesting the presence of augmented central pain processing mechanisms, loss of descending inhibition or a neuropathic pain state; data from (27)). Thus, response to exercise is moderated by other factors such as sensitization of pain processing in the central nervous system. Likely in the latter case, treatment strategies aimed at decreasing the sensitivity of the central nervous system (i.e. desensitizing therapies) are warranted prior to or at least in combination with exercise (37). Psychological factors have also been shown to impede a favourable outcome from exercise programs. For instance, high levels of post-traumatic stress syndrome in patients with chronic WAD predicts ongoing post-traumatic stress following a comprehensive exercise program (2) indicating the need for additional or alternative treatment strategies.

We recently evaluated whether the type of exercise intervention is a determinant of clinically important neck disability or pain reduction in chronic WAD, and whether features of the patient's baseline presentation were associated with outcome following exercise interventions (31). The only significant factor associated with a reduction of both neck pain and neck-related disability at 3 and 12 months, was participation in a specific neck exercise program which was based on a detailed assessment of the patient. Patients allocated to this group had up to 5.3 times higher odds of achieving disability reduction, and 3.9 times higher odds of achieving pain reduction compared to those that participated in general physical activity, even if both groups did have a significant benefit from exercise (31). This result supports the inclusion of exercise as part of the rehabilitation programme even in chronic musculoskeletal pain disorders which have been characterised by a high prevalence of central sensitization.

A systematic review and meta-analysis which evaluated the effects of exercise for low back pain on pain outcome estimated that, from all 43 trials in the review, exercise led to a 3.4 (95% confidence interval [95% CI]=2, 4.7) point reduction in pain, measured on a 0–100 pain scale (20). However, when considering an intervention which involved 1) individually designed exercise programs; 2) supervised home exercises with therapist follow-up, group, and individually supervised exercise delivery strategies; and 3) high-dose or high-intensity exercise programs, multivariable modelling of outcomes demonstrate an expected improvement in pain scores by 18.1 points (95% CI= 11.1 to 25.0). The probability that this represents a clinically important improvement for pain and function outcomes was found to be 29% and 4%, respectively, compared with no treatment, and 3% and 1%, respectively, compared with other conservative treatments (20). However, it is likely that identification of characteristics of the patients who respond best to different types of exercise may substantially enhance the treatment effects.



## **TARGETED EXERCISE INTERVENTIONS FOR MORE EFFECTIVE MANAGEMENT OF SPINAL PAIN**

Although significant and clinically meaningful reductions in pain and disability have been achieved by various exercise programs including low intensity training (focussed on precision and control) and high intensity training (focussed on strength and endurance effects) and biopsychosocially-driven pain management strategies including graded activity, efficacy is likely to be enhanced if the right approach is targeted to the right patient, and within the optimal approach, the individual's characteristics are addressed.

A first step of targeting intervention to the right patient involves determination of which overall exercise approach may be appropriate for an individual. As an example, it is plausible that exercise approaches such as “graded activity” approach may be best suited to an individual where fear avoidance and deconditioning are key elements of the presentation. Conversely, an approach that targets careful modification of motor control to optimise tissue loading would be best suited to an individual whose pain experience continues to include a peripheral contribution related to suboptimal tissue loading. There is preliminary evidence that such a relationship is likely. In a clinical trial we compared exercise targeted at graded activity vs. motor control training for individuals with non-specific low back pain (33). Although both treatments were equally effective when the whole group was considered, the motor control approach was more effective for those who achieved a high score on a specific baseline questionnaire, and graded activity was more effective for those with a low score (34). Although the questionnaire is purported to identify the presence of “instability”, it is more likely that the questionnaire is detecting the contribution of a nociceptive component to the patient's pain driven by physical features of the patient's presentation. This is currently being investigated.

Once it is decided that a motor control approach would be beneficial, it is essential to make informed decisions regarding the best approach to target the individual-specific features of their presentation that relate to symptoms. This requires consideration of the neuromuscular/functional changes induced by a training approach, as these are specific to the mode of exercise performed. For instance, targeted training of the deep neck and back muscles through low intensity repeated voluntary activation of the muscles, increases activation of deep cervical flexor muscles during a cranio-cervical flexion task, improves the speed of their activation when challenged by postural perturbations (neck and back) and enhances the degree of directional specificity of neck muscle activity during multidirectional isometric contractions of the neck. Activity of superficial neck and back muscles can also be reduced with specific motor control training, even after a single session. Importantly, control over the deeper postural muscles was not altered by generic forms of higher intensity exercise even if comparable pain relief was achieved between the low load and specific versus high load and non-specific exercise programme in people with chronic neck pain. Motor cortex representation of the back muscles is modified by specific motor control exercises, but not graded activity. For a review of these specific adjustments to exercise in people with spinal pain, see (14).

In contrast, exercise programs utilizing higher load endurance and strength protocols have shown larger gains in cervical muscle strength, endurance, and resistance to fatigue compared to low load programmes. Further, resistance training targeted at atrophied muscles was required to ameliorate the long-standing atrophy and fatty infiltration in patients with chronic low back pain (see (14) for review). Thus outcomes of training paradigms are specific. It is essential that appropriate paradigms are selected to target the distinct structural and functional

changes in the periphery (e.g. enhanced muscle mass) and across the regions of the nervous system from the spinal cord to the motor cortex and other supraspinal centres that are relevant for the individual patient's symptoms/presentation. In support, recent work highlighted that exercise targeted to specific aspects of neuromuscular function were most effective for those who presented with the neuromuscular impairment to which the exercise was targeted. For example, in patients with low back pain, poor transversus abdominis activation at baseline predicts those who respond best to specific motor control training (15, 44) and specific training of the deep cervical flexor muscles in patients with chronic neck pain reduces pain and increases the activation of these muscles most in patients with the poorest activation of their deep cervical flexors prior to training (13). These findings indicate that a detailed assessment is essential to identify the physical features such as altered neuromuscular function that are likely to be related to the patient's symptoms and that larger improvements are likely if exercise targets to those features. Further large studies are required to corroborate these findings and investigate whether tailoring rehabilitation to the needs of patients can enhance exercise effectiveness. This also implies that assessment of movement and motor behaviours must be enhanced, and screening methods developed/refined to identify patients who are more likely to respond to a specific exercise (compared to an alternative exercise or treatment) and for whom treatment effects are larger. The challenge is to conduct longitudinal studies to confirm or refute the hypothesis that treatment targeted to physical features including neuromuscular dysfunction in the right patients and in a manner that is targeted at the individual patient's needs provides longer term pain relief and ultimately reduces the persistence or recurrence of spinal pain.

## CONCLUSION

There is no doubt that the assessment and management of spinal pain should be considered within a biopsychosocial framework which embraces biological, psychological and social features and their interactions, all of which may contribute to the disorder and to recovery. Yet the relevant contribution of each component will vary for each individual. The large variability in underlying pain mechanism (relevance of nociceptive input or central sensitisation processes) and variability of motor adaptations noted between individuals with neck or low back pain likely contributes to the variable symptomatic benefit experienced by patients following standardized exercise programs. It is not surprising when some studies show little or no effect of exercise interventions in people with spinal pain considering that the people included in the trial may have psychological or social features contributing to their disorder which outweigh physical features that were not addressed with the applied exercise program. Because of this heterogeneity, there can be no recipe approaches and it is likely that better outcomes will be achieved if each patient is regarded as an individual and management programs are designed and tailored to individual's needs. This review has provided evidence of individual adaptations to pain and results from clinical trials which substantiate the hypothesis that exercise outcome will be optimised when targeted to findings of a detailed assessment. This also implies that the diagnostic tests that can be utilised in clinical practice need to be improved notably.

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## FIGURE LEGENDS

**Figure 1.** Optimising management of spinal pain: Outcome of exercise interventions can be optimized when targeted towards the right patients and when tailored to address the presentation and features (including neuromuscular; psychological, social) of each individual's presentation.

**Figure 2A.** A biopsychosocial model of spinal pain. **B.** Weightings of biological, psychological and social features differ between individuals with implications for the relevance of exercise for management. 1. An example where biological factors such as physical features are the main drive maintaining a patient's chronic pain disorder e.g. suboptimal posture/alignment, movement and altered patterns of muscle activation. 2. An example where dominant psychological features such as pain catastrophizing, fear avoidance, anxiety, depression or stress outweigh physical factors. 3. An example where social factors such as poor support or high job demands with low reward are amplifying the patient's perceived pain.

**Figure 3.** High density surface EMG signals detected using a semi-disposable adhesive grid of electrodes over the right upper trapezius muscle as healthy participants performed sustained shoulder abduction. Representative topographical maps (interpolation by a factor 8) of the EMG root mean square value are presented for one subject for the first 5 s of the sustained shoulder abduction contraction performed at baseline and following individual injections of 0.4 ml of hypertonic saline into the cranial and the caudal region of the upper trapezius muscle. Note that regardless of the location of noxious stimulation, the motor adjustment was the same. The greatest reduction of EMG amplitude occurred in the cranial region of the upper trapezius muscle following the injection in either location with a shift of activity towards the caudal region of the muscle. (Reprinted from (8). Copyright © 2009 Elsevier. Used with permission.)

**Figure 4.** A. Area of pain reported following hypertonic saline injection into the right erector spinae (L3) and right medial gastrocnemius muscles. B. Variation between participants in change in EMG amplitude relative to control during pain. Red - percentage of participants with increased EMG, blue - decreased EMG, grey - no change (gray). Similarity of the muscle synergies compared with control. C: Cross-correlation coefficients ( $r$ ) of the muscle synergy activation coefficients between control and LBP, washout LBP, CalfP, washout CalfP, and between LBP and CalfP. [Adapted from (45). Copyright © 2015 The American Physiological Society. Used with permission.]

**Figure 5.** Individual variation in redistribution of muscle activity during acute pain to increase spine protection. A. Electromyography (EMG) recordings were made from 12 trunk muscles. B. Pain-free participants moved slowly forwards and backwards in sitting. C. Pain was induced by injection of hypertonic saline into the longissimus muscle. D. EMG-driven mathematical model was used to estimate spine stability, which increased during pain. E. EMG changes are shown for 12 muscles in 17 participants. Blue - increased EMG during pain, orange - decreased EMG, black - no change. Spine stability increased to protect the spine during pain (Panel D), but was achieved by individual specific patterns of modulation of EMG activity. RA – rectus abdominis; OE – obliquus externus abdominis; OI – obliquus internus abdominis; LD – latissimus dorsi; TES – thoracic erector spinae; LES – lumbar erector spinae; r – right; l – left. [Adapted from (22). Copyright © 2013 John Wiley and Sons. Used with permission.]

**Figure 6.** A. In this study, participants performed multi-directional, multi-planar aiming movements of the head. Nine circular targets (one “central target” plus 8 “peripheral targets”) were placed on a whitewall following a circular trajectory. Participants wore a helmet mounted with laser pointers and the task consisted of moving their head and neck to aim laser pointers

from the central target to each peripheral target following the tempo provided by a metronome. Electromyography (EMG) was recorded from multiple neck muscles. B. The task was completed at baseline (no pain) and immediately following the injection of hypertonic saline into the right splenius capitis muscle (painful condition). C. Mean and SD of the EMG amplitude recorded for each muscle in the painful condition normalized relative to the baseline condition. The gray dotted line indicates the level of activity which would be comparable between conditions. The injected muscle, the right splenius capitis, is highlighted in red; note the overall decreased activity of this muscle. Other muscles showed either an increase or decrease of activity when averaged across all subjects. D. Individual data for each of the eight subjects showing the direction of change in EMG amplitude of each muscle between the baseline and painful condition. Red indicates an increase of EMG amplitude in the painful condition compared to baseline, blue indicates decreased EMG amplitude and white indicates no change. Note the individual specific patterns of modulation of EMG amplitude. No two subjects showed the same strategy. (Right –R and Left – L: Sternohyoid –HYO, Sternocleidomastoid -STER, Anterior Scalene -SCA, Splenius Capitis –SPL, Upper trapezius -UTR, Lower Trapezius -LTR). (Reprinted from (17). Creative Commons.)

**Figure 7.** Mean and standard deviation of the percent reduction in neck pain intensity reported following a rehabilitation program, including specific exercise, for individuals with chronic whiplash associated disorders (WAD) and people with idiopathic neck pain. Note a 47% reduction in neck pain after six weeks of specific exercise in group of patients with mild/moderate idiopathic neck pain. An eight week rehabilitation program including the same

specific exercises resulted in a 37% reduction in neck pain intensity in people with WAD with signs of mechanical hyperalgesia. The response to the same intervention was only 16% in people with WAD with signs of widespread mechanical and cold hyperalgesia. Thus response to exercise is highly variable in people with neck pain disorders and the effect of exercise may be moderated by other factors such as central sensitization.

**Figure 1**

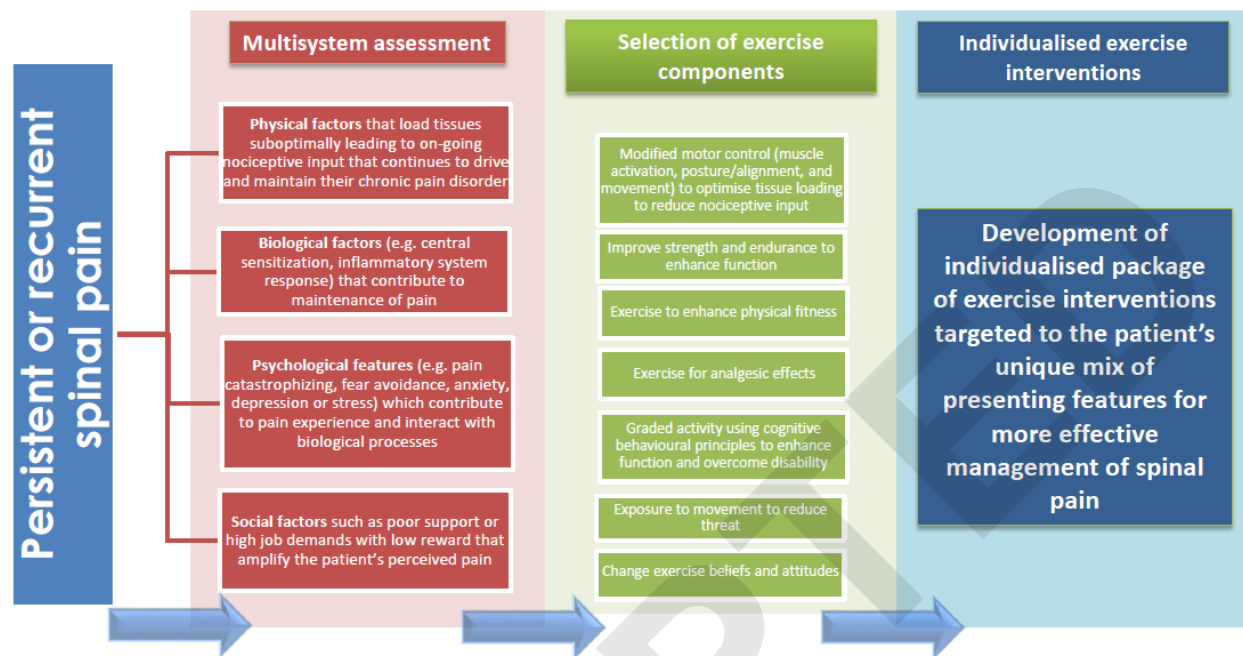
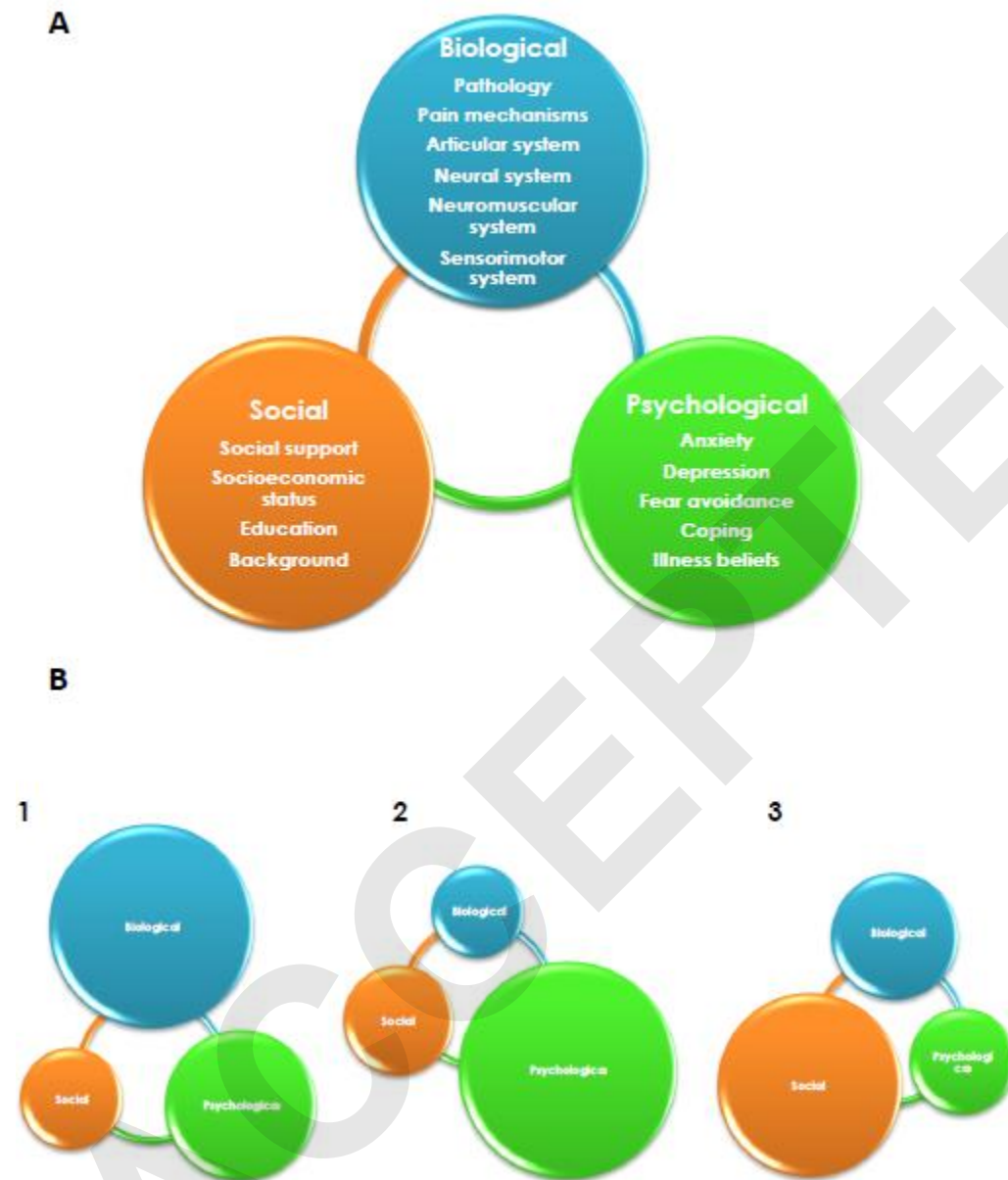


Figure 2





**Figure 3**

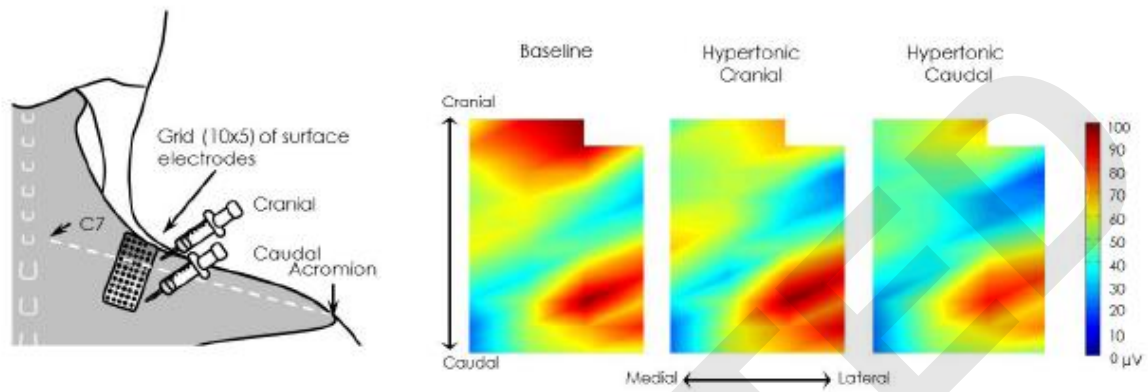


Figure 4

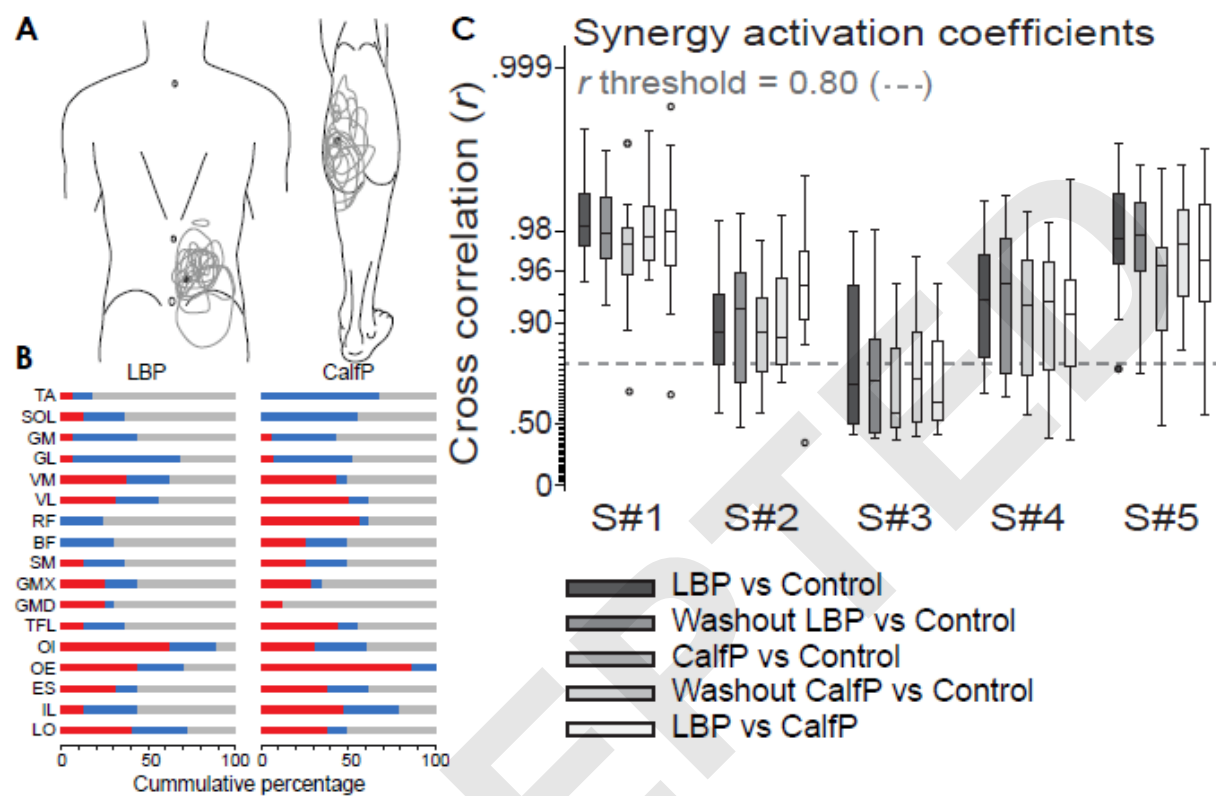


Figure 5

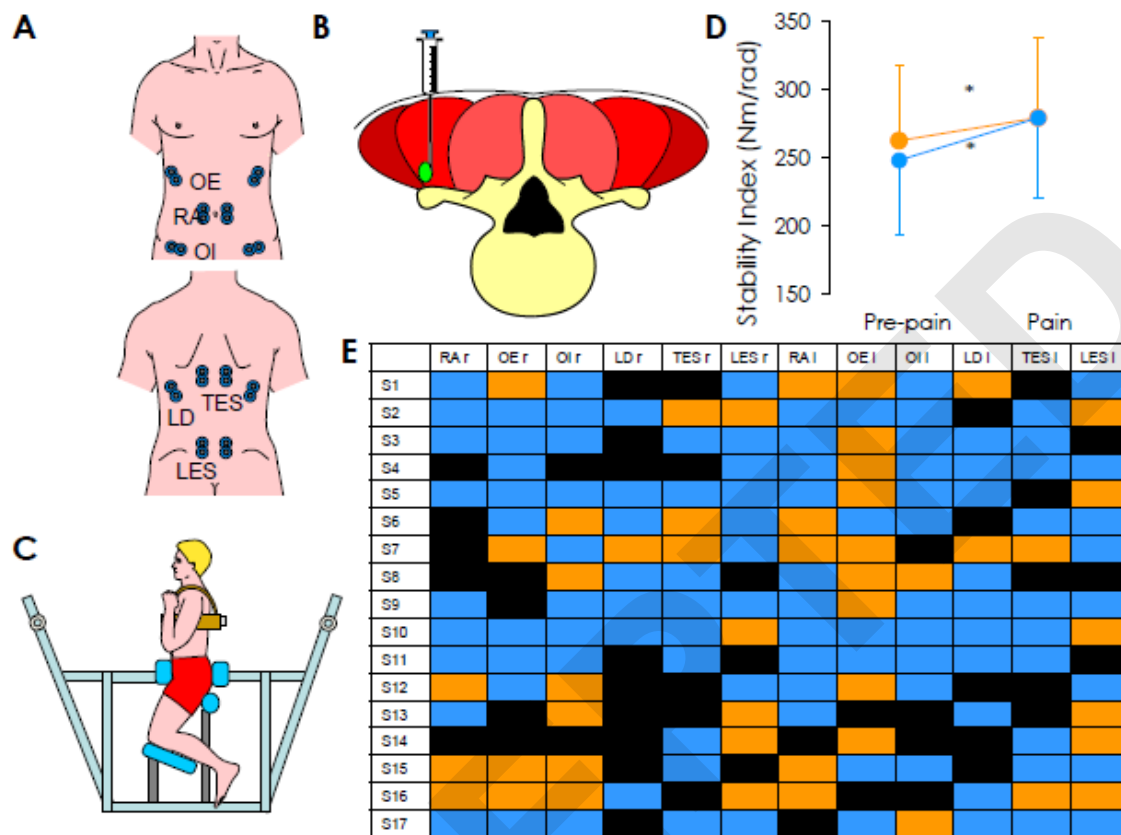
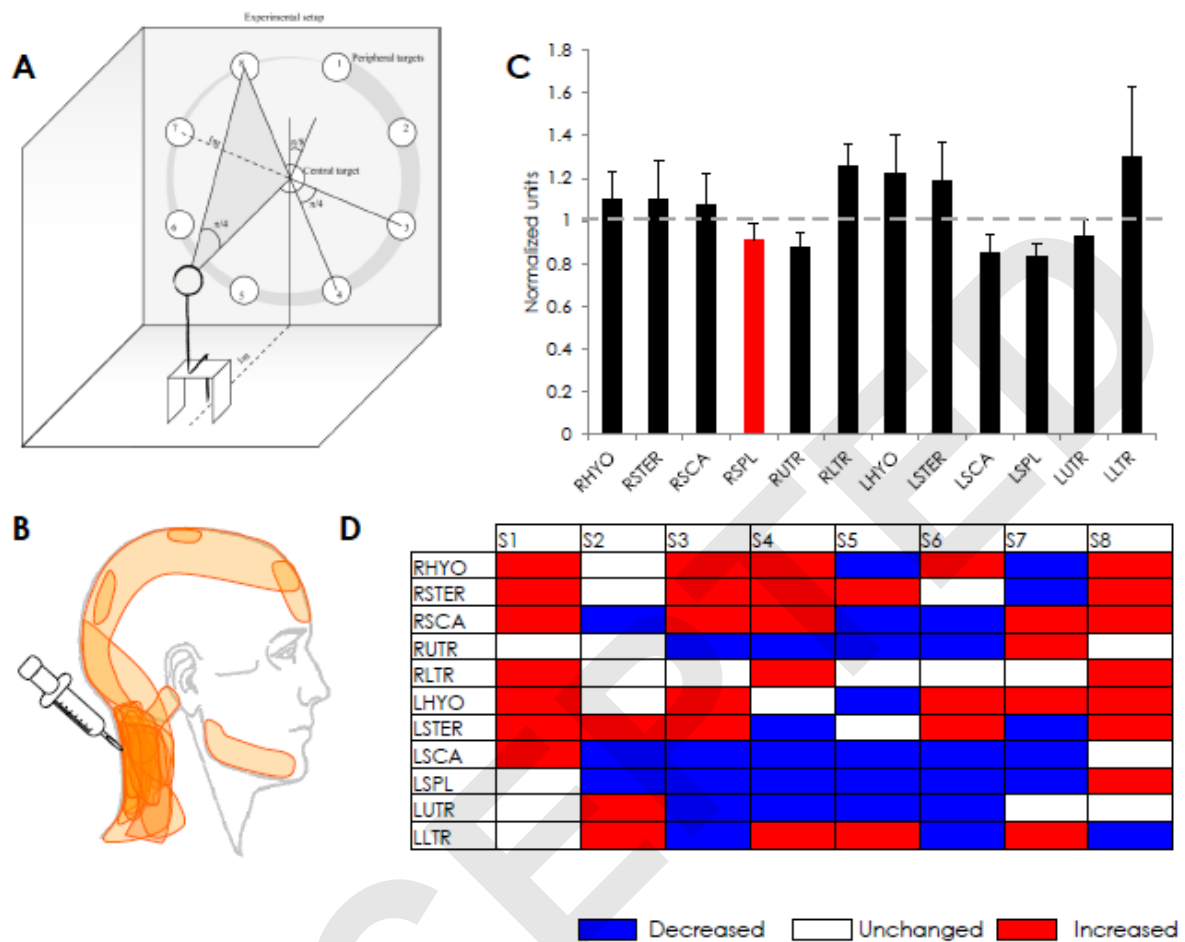


Figure 6



**Figure 7**

